7. When the gonococcus is found with no vaginal discharge or with a very slight discharge, children should also be quarantined, although it is impossible at present to say to what degree such cases may be dangerous in the ward. One of the greatest difficulties in connection with the gonococcus vaginitis arises from the prolonged quarantine rendered necessary, from the fact that these cases are of a very chronic character and very resistant to treatment.

8. The danger to nurses from accidental infection, especially in the eyes, is considerable. At the present time we are not sufficiently instructed in this respect.

OBSTETRICS.

UNDER THE CHARGE OF

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The Pathological Anatomy and Pathogenesis of the Toxæmia of Pregnancy.—EWING, Professor of Pathology in the Medical Department of Cornell University, in the *American Journal of Obstetrics*, February, 1905, publishes a paper recently read before the New York Obstetrical Society.

He has for some time studied the pathology of the hepatic toxæmia of pregnancy and demonstrated the hepatic lesions of three clinical manifestations of the toxæmia of pregnancy. In at least 95 per cent. of all varieties of eclampsia, hemorrhagic hepatitis is found. In fatal cases this lesion is always present. The liver is usually normal in size, reduced in consistence, with many minute hemorrhagic foci in surface and section. Microscopically, a uniform and intense granular hydropic and fatty degeneration of the liver cells, with disorganization of protoplasm, is present. Focal necroses are numerous. There are many minute hemorrhages. This condition is pathognomonic of the disease.

The second is acute yellow atrophy. This is illustrated by the case of a patient, aged twenty-one years, pregnant four and a half months, suffering for two weeks with severe headache, epigastric pain, and vomiting. Emesis gradually ceased, but headache continued, and the patient became disturbed mentally. There was slight fever, epistaxis, jaundice, and muscular twitchings, with one general convulsion two hours before death. The urine was without albumin and casts. Leucin and tyrosin were present.

The liver was slightly reduced in size, its consistence reduced, and on section it was mottled red and yellow. There was complete degeneration of the inner two-thirds of the lobules and intermediate zone of necrotic cells, and a narrow peripheral zone showing slight degeneration.

In the third case the patient had eclampsia, with minimal hepatic lesions. On macroscopic examination the liver was slightly altered. On microscopic examination there was diffuse granular and fatty degeneration, with foci of intense degeneration, partial necrosis, and areas where the cells were distended with pigment. The patient had been pregnant six and a half months and was admitted to the hospital in a stupid condition. She was cyanotic, without jaundice, was delivered of a dead fœtus, had one convulsion, and died in two hours.

These cases show that eclampsia may exhibit hemorrhagic hepatitis, acute yellow atrophy, and autolysis of the liver cells, demonstrable only by microscopic examination.

Ewing also records 3 cases of pernicious vomiting. The first was a well-nourished patient, aged twenty-six years,who had vomiting in the first three months, which subsided, returning in the eighth month. The patient became comatose, and died without convulsions. The liver was anæmic in appearance, and microscopically showed complete necrosis of the inner two-thirds of lobules, with extreme degeneration of the outer third.

Case 2 was a well-nourished woman, aged thirty-two years, who had suffered severely from nausea in former pregnancies. In the third severe vomiting subsided in the second month, but began again severely in the eighth. Treated as hysterical, and vomiting controlled for two days by mental influence. Died on the eighth day of the attack, without convulsions or jaundice. The liver was very soft and mottled red and yellow, with intense granular hydropic degeneration of the liver cells.

Case 3 was that of a woman brought to the hospital by her husband, who stated that for a week past she had been acting strangely and vomiting repeatedly, which was attributed to alcoholism. Was transferred with a diagnosis of hysteria and fibroids. Found to be pregnant and suffering from toxæmia. Death occurred twenty-four hours later. The liver was very soft, congested and fatty, and the blood remarkably thick and cohesive, and there was intense degeneration of liver cells.

It is interesting to note that the hepatic lesions of these cases of pernicious vomiting are identical with those found in eclampsia.

Similar lesions are observed among animals. A large rabbit in the animal house of the Cornell Laboratory gave birth to a litter of five and soon after died. The liver was extremely fatty with extensive degeneration. No other cause for death was found. Ten c.c. of blood from the cadaver of one of the cases of pernicious vomiting was injected into the peritoneal cavity of a rabbit. The animal had muscular spasms, seemed moribund, but recovered. Five days later it died, the liver in a condition of extreme degeneration. An injection of 10 c.c. of normal human blood into the peritoneal cavity of a rabbit rendered the animal somewhat restless for about thirty minutes, after which there were no further symptoms.

The author also reports a case of acute yellow atrophy of the liver occurring after abortion at three months. The patient never recovered, but suffered from indigestion, vomiting, and abdominal pain. Death occurred on the fourteenth day. The liver was very soft, uniformly red; its lobules obliterated. Microscopically, it was found to be in the late stage of yellow atrophy. He also reports 2 cases of acute leukæmia following pregnancy, terminating fatally, in which the lesions of acute yellow atrophy, with extensive necrosis, were found in the liver.

Ewing believes that a failure of the oxidizing capacity of the liver produces the disturbance of nitrogenous metabolism, causing the clinical manifestations of the toxæmia of pregnancy. The proteid derivatives normally combined by the liver into urea are no longer combined, but circulate free in the blood in a poisonous form, and are partially excreted by the kidneys. Other proteid derivatives, as those containing sulphur, also fail to be oxidized and contribute to the toxæmia. Large quantities of leucin, tyrocin, and lycin have been found in the blood of acute yellow atrophy. The complex nature of the sources of these poisons makes less obscure the fact that the clinical manifestations of the toxæmia of pregnancy vary from mild vomiting to acute yellow atrophy. Recent study shows us that these morbid processes in the liver are common, and, when detected, and the patient is given proper hygienic care, are often followed by recovery. Pathology teaches us that we cannot separate the mild and fatal cases from the standpoint of etiology of the vomiting of pregnancy. Pernicious nausea of pregnancy is a very different thing from occasional vomiting, and is attended with the failure of urea formation, which is characteristic.

In the urine of the toxæmia of pregnancy we find evidence of deficient oxidation of proteid derivatives. Instead of urea, we find those substances whose oxidation should result in urea. The hypobromite test for urea is not considered infallible because leucin, tyrosin, and ammonium are estimated with urea by this test. Ewing believes that the examination for unoxidized proteid derivatives will be of value in estimating the seriousness of the case. The urine should be freed from albumin and the filtrate evaporated until the characteristic crystals can be identified by the microscope. Albumin and casts are sometimes present, but may be absent in dangerous stages of the disease and even in fatal cases.

The absence of jaundice is seen in fatal cases of nausea of pregnancy with the hepatic lesions of yellow atrophy. In these cases the bile-producing function of the liver is inhibited.

At present we believe that the toxæmia of pregnancy results from functional disturbance of the liver, usually attended by severe anatomical lesions and secondarily by functional and anatomical disturbance in the kidneys and other organs. The production of urea is exclusively a function of the liver. While disturbance of the kidneys exists from the first, it only becomes pronounced when poisons resulting from failure of oxidation cause degeneration, congestion, and exudative inflammation in these organs. Hence, the disease may be far advanced before albuminuria appears. As the disease is a functional disturbance, we find some fatal cases with minimal lesions of the liver. The part which the thyroid gland plays in the morbid process is worthy of consideration in studying this question.

In one of these subjects the intestinal tract was found greatly distended with saline solution, and the patient had failed to absorb the fluid. In other cases the blood was found remarkably thick, viscous, and cohesive. While saline irrigation is indicated it cannot always be relied upon in patients incapable of absorbing fluids.

Again, the absence of the necessarily fatal character in the disease follows from the pathological study. If elimination can be maintained and production of the poison prevented, there is nothing to prevent the continuance of the patient's life. There is no uncontrollable infection, nor is there a hopeless anatomical lesion. Saline irrigation and infusion are especially valuable, and Ringer's fluid, composed of sodium chloride, calcium chloride, potassium chloride, and sodium bicarbonate, is especially valuable.

Instrumental Treatment in Post-partum Hemorrhage from Uterine Atony.—In the *Zentralblatt für Gynäkologie*, 1905, No. 9, is described an instrument devised by BUMM for checking post-partum hemorrhage. It consists of a metal ball covered with sterile gauze or with a sterile towel, and inserted into the uterus. The uterus is then massaged, and the counterpressure of the body within the womb checks the hemorrhage.

Antistreptococcic Serum in the Treatment of Puerperal Sepsis.—BOECK (*Zentralblatt für Gynäkologie*, 1905, No. 4) has treated 20 cases of puerperal sepsis with antistreptococcic serum. In 5 of these Marmorek's serum was employed, in 12 Aronsohn's serum, and in 3 a new serum. Streptococci were present in 13 cases, saprophytes in 2, mixed infection in 1, and in 4 cases no bacteriological examination was made. In the 13 cases having streptococci the serum produced an evident effect upon the pulse and temperature in 5. There was a doubtful result in 6, and in 2 its use was without effect. It was also useless in cases infected with saprophytes, but was beneficial to the patient in mixed infection. In the cases not examined bacteriologically, 1 seemed much benefited by the serum, 1 was possibly improved, and in 2 no result could be observed.

GYNECOLOGY.

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Arteriosclerosis as a Cause of Abdominal Pain.—BUCH (*St. Petersburger med. Wochenschrift*, 1904, No. 27), from an analysis of 14 cases of his own and many recorded by other observers, concludes that so-called "splanchnic" arteriosclerosis, or disease of the vessels within the abdominal cavity, can be diagnosed as certainly as can the same condition in the brain or extremities. The symptoms are liable to be mistaken for the dyspepsia accompanying cardiac affections, as well as cancer or other diseases of the stomach and intestines, as the elderly patients have severe attacks of pain in the epigastrium. It is important to note that in arteriosclerosis the pains are increased on exertion and